17. (Amended) A method for inhibiting the binding of IgE to its high-affinity IgE receptor *in vivo* comprising administering to a patient in need thereof a composition comprising an adenoviral vector comprising a nucleic acid encoding an anti-IgE antibody or IgE binding fragment thereof.

18. (Canceled)

19. (Amended) The method of claim 17 [[18]], wherein the anti-IgE antibody is Hu901.

20. (Canceled)

21. (Amended) The method of claim 24 [[20]], wherein nucleic acid encodes an scFv of Hu901.

22. (Amended) A method of suppressing or attenuating an IgE-mediated allergic disease *in vivo* comprising administering to a patient in need thereof a composition comprising [[a]] an adenoviral vector comprising a nucleic acid encoding an anti-IgE antibody or IgE-binding fragment thereof, wherein said anti-IgE antibody inhibits or blocks the binding of IgE to the high affinity IgE receptor.

23. (Amended) The method of any one of claims 17, 19, 21 or 22, wherein the ~~vector is an~~ adenoviral vector ~~with~~ comprises a human cytomegalo virus promoter for expression of the anti-IgE antibody.

24. (NEW) The method of claim 17, wherein the adenoviral vector encodes a single-chain Fv.

25. (NEW) A method of producing an anti-IgE antibody *in vivo* comprising administering to a patient in need thereof a composition comprising an adenoviral vector comprising a nucleic acid encoding an anti-IgE antibody or IgE binding fragment thereof.

26. (NEW) The method of claim 25, wherein the anti-IgE antibody is TES-C21.

27. (NEW) The method of claim 26, wherein the TES-C21 antibody has been humanized.

REMARKS

Applicant has amended the claims to more particularly and distinctly claim that which Applicant regards as his invention. Claims 18 and 20 have been canceled without prejudice or disclaimer to the subject matter contained therein. Applicant has added new claims 24-27. No new matter has been introduced by these amendments.

I. Rejection under 35 U.S.C. § 112, First Paragraph

Claims 17-23 have been rejected as lacking enablement for *in vivo* methods of inhibiting the binding of IgE to the high affinity IgE receptor for any vector. Applicants respectfully traverse this rejection in view of the amendments and the following remarks.

It has been well documented, both in animal models as well as humans, that administering anti-IgE antibodies that interfere with the binding of IgE to the high affinity IgE receptor reduces and/or prevents the release of mediators that